

Long-term histologic and electrophysiologic evaluation of the alloy retinal tack*

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Abstract. The retinal tack is a useful adjunctive instrument in the repair of complicated retinal detachments. To examine the tissue response of chronically implanted alloy tacks, we implanted a series of tacks into rabbit eyes. Their effects were observed at 1 and 4 weeks, at 6 months, and at 2.5 years after insertion. Our observations indicate that the histology and electrophysiology of the retina was generally not affected. All tacks were surrounded by connective and/or glial tissue scar and induced firm retinal adherence. The retinal architecture was normal within 1 mm of the scar, indicating that these alloy tacks do not cause tissue damage outside the immediate area of the wound, even when left in place over a period of 2 years.

Introduction

Mechanical retinal fixation is a useful adjunctive technique in the repair of complicated retinal detachments [1, 2, 4, 5]. The purpose of retinal tacks is to provide temporary intraoperative fixation of the retina to the wall of the eye so as to facilitate the repair of complicated retinal detachments. Prolonged retinal fixation may be beneficial in some cases. Although detailed histologic evaluation has been done on the effects of titanium retinal tacks on ocular tissue, no such study is available for the widely used alloy tack [4, 5]. To examine the tissue response to chronically implanted alloy tacks, we implanted a series of tacks into rabbit eyes. The eyes were studied using serial electroretinography (ERG), followed by light and electron microscopy and autoradiography of the retina, choroid, and other ocular tissues. We found that the alloy tacks caused minimal injury to the retina and ocular structures.

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Materials and methods

Pigmented rabbits (2.5–3.5 kg) were used in these studies. A total of 22 animals were anesthetized with intramuscular injections of ketamine hydrochloride (30 mg/kg) and xylazine hydrochloride (5 mg/kg), 0.5 ml of retrobulbarly injected 2% lidocaine, and topical 0.5% proparacaine. Mydriasis was produced by topical applications of 5% phenylephrine hydrochloride and 0.25% tropicamide. Both eyes were subjected to transcleral cryotherapy 4–8 mm posterior to the corneoscleral limbus and nasally and temporally 180° degrees apart. After 1 month, the rabbits underwent baseline ERG as described below.

ERG data for all groups were gathered by Ganzfeld photostimulation (stimulus intensity, 1.74 foot lambert). We obtained ERGs from each rabbit both before and 1 month after cryoretinopathy and at 7 and 28 days, 6 months, and 2.5 years after tack insertion. At every interval, each rabbit was anesthetized as described, except for the retrobulbar anesthesia, and examined by indirect ophthalmoscopy in both fundi. We used topical applications of 5% phenylephrine hydrochloride and 0.25% tropicamide to dilate each pupil and 0.5% proparacaine to anesthetize the corneas. Burian-Allen contact lens electrodes lined with methylcellulose gel were attached to both corneas and a common ground electrode was placed subdermally in the scalp. The rabbit's head with attached electrodes was surrounded by a Ganzfeld photostimulator, and 3 min light adaptation was followed by 11 min dark adaptation. Both eyes then received three exposures at 1-min intervals and the ERG responses were simultaneously recorded on paper. We measured the b-wave amplitudes for each test stimulus in each eye. An average value was calculated for eyes with tacks and the result was expressed as the ratio of their b-wave amplitudes (experimental value/control value).

The tacks consisted of alloy previously approved by the United States Federal Drug Administration (FDA) for surgical implantation: cobalt-nickel-chromium-molybdenum-tungsten-iron. Tack insertion was performed in the left eye of each rabbit, with the right eye serving as a control (no operation).

We divided the rabbits into two groups. In the first group (18 rabbits), a sclerotomy incision was made in the most posterior area of the superior temporal site of the cryo-treated retina, through which the retinal tack was inserted, engaged by the tack inserter. The tack was passed through the formed vitreous and inserted into the retina, choroid and sclera, usually 3–4 mm posterior to the medullary ray at the posterior pole. Two tacks were placed in each of the 18 eyes: one under the nasal wing and the other under the temporal wing. Eyes were examined by indirect

ophthalmoscopy immediately after tack insertion and then at intervals until the animals were killed. In the second group of animals (4 rabbits), a central vitrectomy was performed using an Ocutome vitreous cutter and the retinal tacks were then inserted as described above.

At 2 h before the animals were killed, control and tack-containing eyes from selected animals (6-month group) were given a mid-vitreous injection of tritiated thymidine to label cells preparing for division. After fixation in 10% buffered formaldehyde solution containing 3% glutaraldehyde with 0.1 M cacodylate buffer (pH 7.3), pieces of posterior and peripheral retina were embedded in paraffin for light microscopy and in Spurr low-viscosity embedding medium for electron microscopy.

Both eyes from each rabbit were enucleated immediately after the animal's death and then placed directly into 3% glutaraldehyde containing 0.1 M cacodylate buffer (pH 7.3). For eyes containing tacks, tissue pieces were prepared from regions that lacked tacks. Tack sites were isolated with a razor and processed for embedding in plastic following fixation in 3% glutaraldehyde and postfixation in 2% osmium tetroxide. The tacks were removed from processed tissue before embedment. Paraffin-embedded tissues were sectioned (5 μ m) and stained with hematoxylin and eosin. Ultrathin sections (70–90 nm) were stained with uranyl acetate and lead citrate and then examined in a transmission electron microscope.

Autoradiographs were prepared by dipping the sections in liquid emulsion, followed by an exposure period of 6 days for paraffin sections and 30 days for plastic sections. Positive controls were run concurrently.

Results

Two retinal tacks were implanted at the temporal and nasal sides near the medullary wing in one eye. Of 28 tacks, 7 were dislodged during the 1st week, although they had initially been substantially inserted in the wall of the eye. Of 24 tacks, 12 became displaced after 4 weeks. Of 22 tacks, 15 were dislodged at 6 months. In the vitrectomy group, none of the tacks had been displaced by the end of 1 week; however, one of six tacks were dislodged at 4 weeks, and one of four became displaced at 6 months.

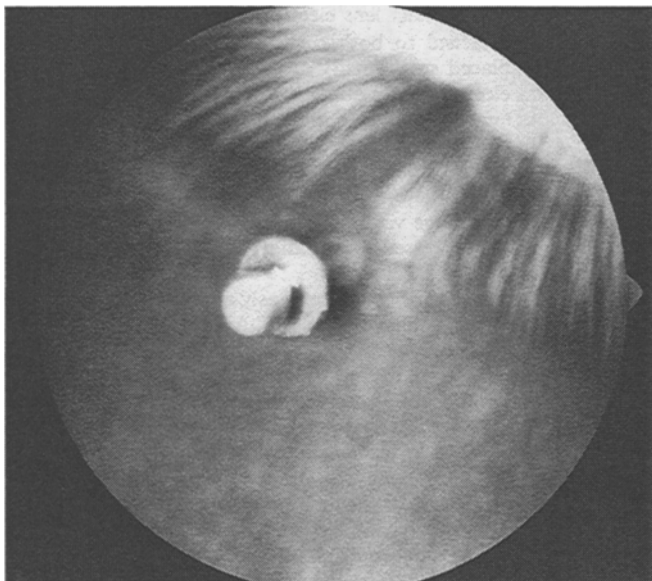


Fig. 1. Rabbit retina at 2.5 years after tack insertion. Note the lack of apparent findings

Small preretinal and vitreous hemorrhages around the tacks were observed in some eyes 1 week later. In some cases, the tack loosened from the retina 1 week after surgery. A flat, white scar occurred at the perforation site. We also observed the formation of vitreous strands at the head of one tack, connecting the other tack at the insertion site. After 4 weeks, the preretinal and vitreous hemorrhages had been absorbed in almost

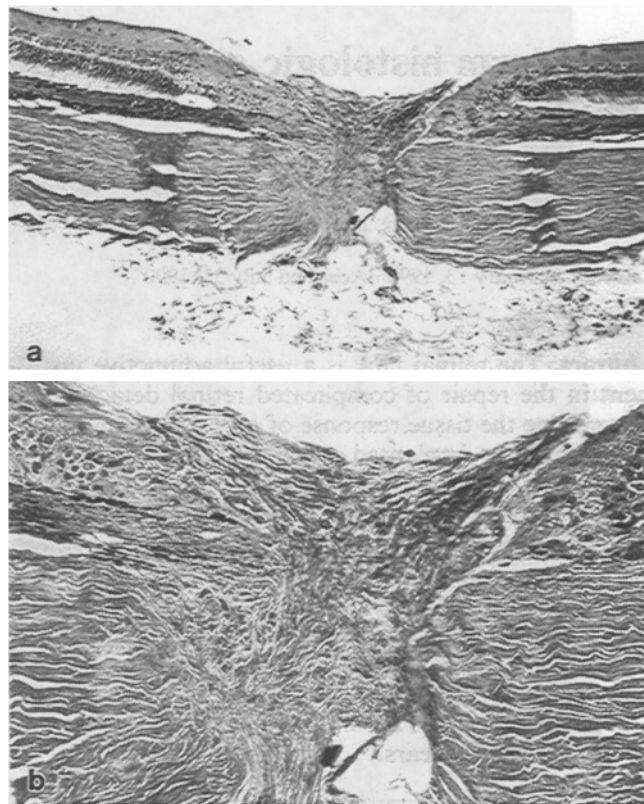


Fig. 2. a Light micrograph of the retina near a tack at 6 months after insertion, $\times 100$. b Higher magnification of Fig. 4. Note that the outer nuclear layer is directly attached to the retinal pigment epithelial cell layers. The retinal tissue was replaced by fibrous scar. $\times 400$

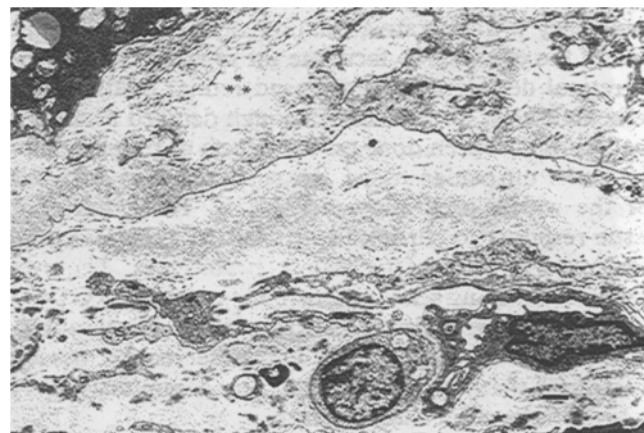


Fig. 3. Electron micrograph of the scar tissue surrounding a tack near Bruch's membrane at 2.5 years after insertion. Note the collagen deposition in the sub-RPE layer (*asterisk*) as well as in the outer part of Bruch's membrane. Bar = 1 μ m

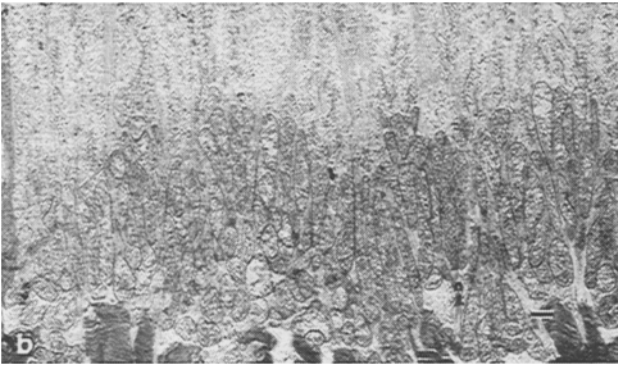
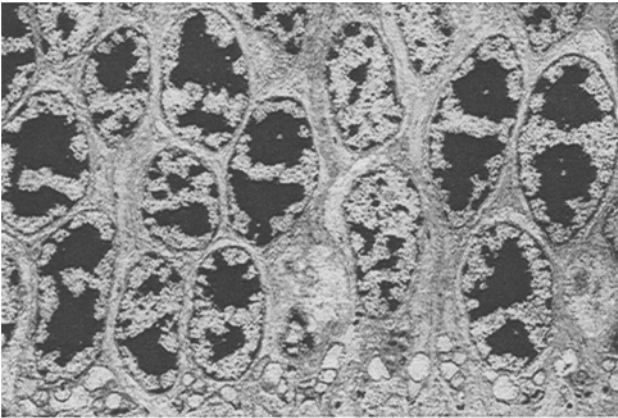
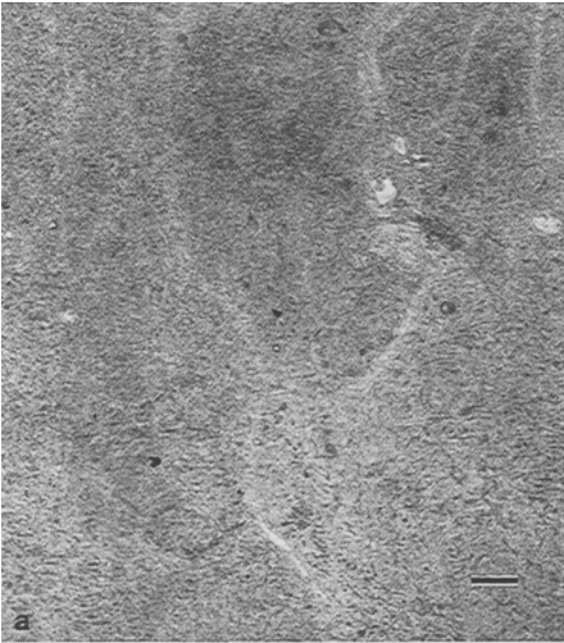


Fig. 4. **a** Electron micrograph of normal inner retina adjacent to a tack site at 2.5 years after insertion. Note the normal appearance of the internal limiting membrane and nerve-fiber layer. Bar = 1 μ m. **b** Electron micrograph of normal outer retina adjacent to a tack site at 2.5 years after insertion. Note the normal appearance of photoreceptor cell layers. Bar = 1 μ m

all eyes. We noticed a white scar and pigmentation around the tacks at this stage. Some of the tacks became partially dislodged due to vitreous traction. There were no apparent vitreous or abnormal retinal findings in rabbit eyes at 2.5 years after tack implantation (Fig. 1).

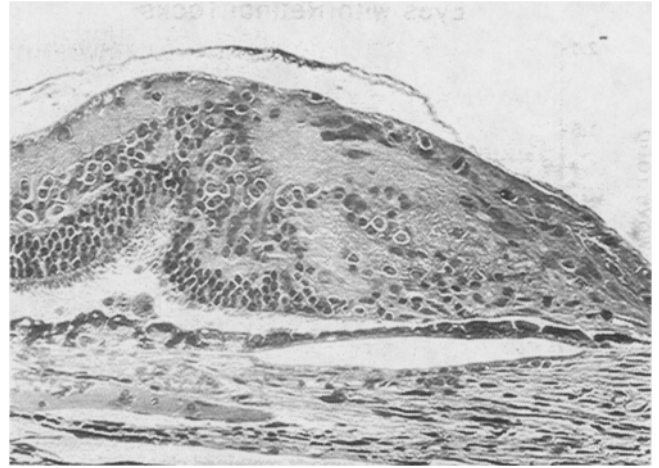


Fig. 5. Light micrograph of a rabbit retina near the tack at 6 months after insertion. Macrophages can be seen in the subretinal spaces. $\times 250$

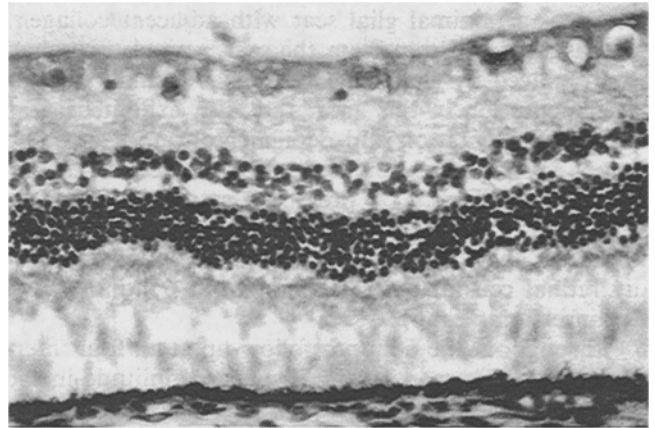


Fig. 6. Autoradiograph of a rabbit retina at 6 months after tack insertion. Note the absence of radiolabel. $\times 250$

The area around the tack was sharply delimited from the adjacent normal retina. Inner and outer segments of photoreceptor cells were absent at the site of tack insertion (Fig. 2), and the outer nuclear layer was directly attached to the retinal pigment epithelial (RPE) cell layers in this area, resulting in the fusion of tissue to RPE cell layers.

Photoreceptor cell layers were replaced by glial cells. Muller and RPE cells were juxtaposed around the site of tack insertion. In some areas around the tacks, endothelial cells lining the choriocapillaries were seen and Bruch's membrane appeared to be slightly irregular. A defect in the basal lamina of RPE cells was observed, with surrounding collagen fibers (Fig. 3). The choroid was thickened near the tack site, and the choroidal vessels appeared to be congested. The scar tissue surrounding the tacks was avascular. Apart from the tack scar, the retinal structure appeared to be normal (Fig. 4). The underlying RPE cells and choroidal melanocytes appeared to be normal. Posterior vitreous collagen was attached to the retinal surface in this portion. The scar

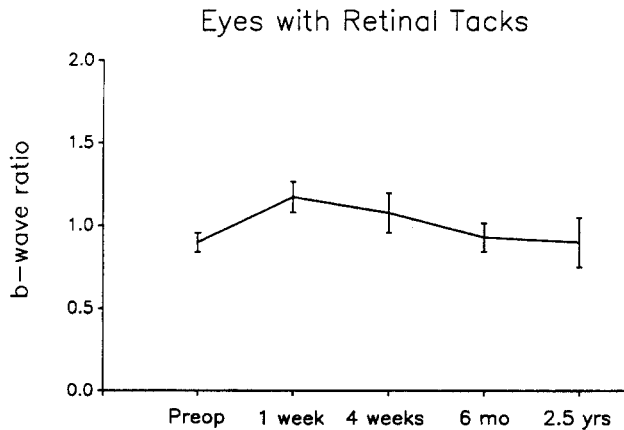


Fig. 7. Electroretinogram. Note that the ratio of b-wave amplitude in the left eyes vs right eyes of rabbits remained close to 1

tissue appeared to be derived from the sclera and choroid.

About 0.5 mm away from the center of the tack scar, there was a minimal glial scar with adjacent collagen; at a distance of 1 mm from the scar, retinal tissue had been replaced by glial cells. There was a small amount of pigmentation in this area; the RPE exhibited areas of hyperpigmentation and depigmentation in the same region. Macrophages could also be identified in the sub-retinal space (Fig. 5). A preretinal membrane that appeared to be glial in nature formed at the vitreoretinal interface. There was no uptake of [^3H]-thymidine by any retinal cells at the insertion site at 6 months after tack insertion (Fig. 6).

Throughout the 6-month follow-up at all four intervals tested, the mean ratio of b-wave amplitude in the left eyes vs right eyes of rabbits remained close to 1 (Fig. 7). The value for one rabbit that had undergone tack insertion 2.5 years previously was also close to 1.

Discussion

The effects of retinal tacks were observed at 1 and 4 weeks, at 6 months, and at 2.5 years after tack insertion. Our observations showed that the histology and electrophysiology of the retina was generally not affected by the tacks. All tacks were surrounded by connective tissue and/or glial scars. The retinal architecture was normal within a distance of 1 mm from the scar, suggesting that the tacks did not cause significant tissue damage outside the immediate site of their insertion. The tacks used in this study had a sharp cutting tip that penetrated the sclera without deforming the globe [4, 5]. The wound site healed well in the eyes studied, with minimal tissue reaction being evident; autoradiography confirmed this impression.

The mean ratio of b-wave amplitude in the left eyes vs right eyes of rabbits remained close to 1. This ratio

did not deteriorate with time, indicating that the composition of the tack is biocompatible with the retina and has a low level of toxicity. However, because of the short period of dark adaptation and the fact that there were no special arrangements for recording an isolated ERG of the rod system, slight changes in rod-system function after tack insertion cannot be fully ruled out [6–8].

The scar tissue consisted of glial and connective tissue, probably deriving from the retina, choroid, and sclera. However, the RPE cells seemed to make only a small contribution to the scar tissue surrounding the tacks because there was little pigment in the scar tissue [3].

Some tacks became dislodged during periods lasting from 1 week to 6 months after their insertion; we believe that this was due to the extremely thin sclera of the rabbit, as it appears to occur much less frequently in human eyes (<1%) in which posterior vitreous detachment (PVD) has developed or in cases in which a vitrectomy precedes tack implantation [5]. Based on these findings, however, we do not recommend the use of retinal tacks without a PVD or vitrectomy.

Electron microscopy of the tack site demonstrated the lack of toxicity to the retina outside the immediate area of scarring induced by the tack. The neuronal and glial-cell elements remained normal in appearance up to the 2.5-year follow-up. The surgical wound was small and the scar, limited.

We observed a close, well-developed approximation between Muller cells and RPE cells around the site of tack insertion; simultaneously we observed a minimal tissue response to the tacks. This study indicates that alloy tacks can be used to secure the retina and do not appear to be toxic when chronically implanted in the eye.

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